What is claimed:

1. A method to protect a mammal from airway hyperresponsiveness and/or airflow limitation associated with a respiratory disease involving an inflammatory response, comprising administering to said mammal a TGFβ-regulating agent selected from the group consisting of a pan-specific TGFβ-inhibiting agent, a TGFβ1-stimulating agent, TGFβ1, a TGFβ2-inhibiting agent, a TGFβ3-inhibiting agent, and combinations thereof.

2. The method of Claim 1, wherein said TGFβ-regulating agent is an antibody.

3. The method of Claim 2, wherein said antibody is selected from the group consisting of a pan-specific TGFβ antibody, a TGFβ2-specific antibody, a TGFβ3-specific antibody, a pan-specific TGFβ receptor-specific antibody, a TGFβ1 receptor-specific antibody, a TGFβ2 receptor-specific antibody and a TGFβ3 receptor-specific antibody.

4. The method of Claim 1, wherein said TGFβ-regulating agent is an antisense oligonucleotide.

5. The method of Claim 4, wherein said antisense oligonucleotide hybridizes under stringent hybridization conditions to a nucleic acid molecule encoding a protein selected from the group consisting of TGFβ2 and TGFβ3.

6. The method of Claim 1, wherein said TGFβ-regulating agent is a TGFβ-specific ribozyme.

7. The method of Claim 1, wherein said TGFβ-regulating agent is a TGFβ receptor agonist.

8. The method of Claim 1, wherein said TGFβ-regulating agent is a TGFβ receptor antagonist.

9. The method of Claim 1, wherein said TGFβ-regulating agent is an isolated TGFβ1 protein.

10. The method of Claim 1, wherein said TGFβ-regulating agent is an isolated nucleic acid molecule encoding a TGFβ1 protein, wherein said nucleic acid molecule is operatively linked to a transcription control sequence.

11. The method of Claim 10, wherein said isolated nucleic acid molecule is administered to said mammal complexed with a liposome delivery vehicle.

12. The method of Claim 10, wherein said isolated nucleic acid molecule is administered to said mammal in a viral vector delivery vehicle.

13. The method of Claim 12, wherein said viral vector delivery vehicle is from adenovirus.

14. The method of Claim 10, wherein said isolated nucleic acid molecule, when administered to said mammal, is expressed in cells of said mammal.

15. The method of Claim 1, wherein said disease is a chronic obstructive pulmonary disease of the airways.

16. The method of Claim 1, wherein said disease is selected from the group consisting of asthma, allergic

bronchopulmonary aspergillosis, hypersensitivity pneumonia, eosinophilic pneumonia, emphysema, bronchitis, allergic bronchitis bronchiectasis, cystic fibrosis, tuberculosis, hypersensitivity pneumotitis, occupational asthma, sarcoid, reactive airway disease syndrome, interstitial lung disease, hyper-eosinophilic syndrome, rhinitis, sinusitis, and parasitic lung disease.

17. The method of Claim 1, wherein said disease is selected from the group consisting of asthma, occupational asthma and reactive airway disease syndrome.

18. The method of Claim 1, wherein said TGFβ-regulating agent is administered by at least one route selected from the group consisting of oral, nasal, topical, inhaled, transdermal, rectal and parenteral routes.

19. The method of Claim 1, wherein said TGFβ-regulating agent is administered by a route selected from the group consisting of intramuscular, subcutaneous, inhaled and nasal routes.

20. The method of Claim 1, wherein administration of said TGFβ-regulating agent reduces airway hyperresponsiveness in said mammal.

21. The method of Claim 1, wherein said TGFβ-regulating agent decreases methacholine responsiveness in said mammal.

22. The method of Claim 1, wherein said TGFβ-regulating agent decreases airways fibroproliferation in said mammal.

23. The method of Claim 1, wherein said TGFβ-regulating agent decreases lung inflammation in said mammal.

24. The method of Claim 1, wherein said TGFβ-regulating agent reduces the airflow limitation of a mammal such that the FEV_1/FVC value of said mammal is improved by at least about 5%.

25. The method of Claim 1, wherein administration of said TGFβ-regulating agent results in an improvement in a mammal's $PC_{20methacholine}FEV_1$ value such that the $PC_{20methacholine}FEV_1$ value obtained before administration of the TGFβ-regulating agent when the mammal is provoked with a first concentration of methacholine is the same as the $PC_{20methacholine}FEV_1$ value obtained after administration of the TGFβ-regulating agent when the mammal is provoked with double the amount of the first concentration of methacholine.

26. The method of Claim 24, wherein said first concentration of methacholine is between about 0.01 mg/ml and about 8 mg/ml.

27. The method of Claim 1, wherein said TGFβ-regulating agent is administered in an amount between about 0.1 microgram x kilogram^{-1} and about 10 milligram x kilogram^{-1} body weight of a mammal.

28. The method of Claim 1, wherein said TGFβ-regulating agent is administered in a pharmaceutically acceptable excipient.

29. The method of Claim 1, wherein said mammal is a human.

30. A method for protecting a mammal from airways fibrosis associated with a respiratory disease involving inflammation, comprising administering to said mammal a TGFβ-regulating agent selected from the group consisting of a pan-specific TGFβ-inhibiting agent, a TGFβ1-stimulating agent, TGFβ1, a TGFβ2-inhibiting agent, a TGFβ3-inhibiting agent, and combinations thereof.

31. A method for prescribing treatment for airway hyperresponsiveness and/or airflow limitation associated with a respiratory disease involving an inflammatory response, comprising:

a) administering to a mammal a TGFβ-regulating agent selected from the group consisting of a pan-specific TGFβ-inhibiting agent, a TGFβ1-stimulating agent, TGFβ1, a TGFβ2-inhibiting agent, a TGFβ3-inhibiting agent, and combinations thereof;

b) measuring a change in lung function in response to a provoking agent in said mammal to determine if said TGFβ-regulating agent is capable of modulating airway hyperresponsiveness; and

c) prescribing a pharmacological therapy comprising administration of TGFβ-regulating agent to said mammal effective to reduce inflammation based upon said changes in lung function.

32. The method of Claim 31, wherein said provoking agent is selected from the group consisting of a direct and an indirect stimuli.

33. The method of Claim 31, wherein said provoking agent is selected from the group consisting of an allergen, methacholine, a histamine, a leukotriene, saline, hyperventilation, exercise, sulfur dioxide, adenosine, propranolol, cold air, an antigen, bradykinin, acetylcholine,

a prostaglandin, ozone, environmental air pollutants and mixtures thereof.

34. The method of Claim 31, wherein said step of measuring comprises measuring a value selected from the group consisting of FEV_1, FEV_1/FVC, $PC_{20methacholine}FEV_1$, post-enhanced pause (Penh), conductance, dynamic compliance, lung resistance (R_L), airway pressure time index (APTI), and peak flow.

35. A formulation for protecting a mammal from a disease involving inflammation, comprising a TGFβ-regulating agent selected from the group consisting of a pan-specific TGFβ-inhibiting agent, a TGFβ1-stimulating agent, TGFβ1, a TGFβ2-inhibiting agent, a TGFβ3-inhibiting agent, and combinations thereof, and an anti-inflammatory agent.

36. The formulation of Claim 35, wherein said anti-inflammatory agent is selected from the group consisting of an antigen, an allergen, a hapten, proinflammatory cytokine antagonists, proinflammatory cytokine receptor antagonists, anti-CD23, anti-IgE, anticholinergics, immunomodulating drugs, leukotriene synthesis inhibitors, leukotriene receptor antagonists, glucocorticosteroids, steroid chemical derivatives, anti-cyclooxygenase agents, anti-cholinergic agents, beta-adrenergic agonists, methylxanthines, anti-histamines, cromones, zyleuton, anti-CD4 reagents, anti-IL-5 reagents, surfactants, anti-thromboxane reagents, anti-serotonin reagents, ketotiphen, cytoxin, cyclosporin, methotrexate, macrolide antibiotics, heparin, low molecular weight heparin, and mixtures thereof.

37. The formulation of Claim 35, wherein said formulation comprises a pharmaceutically acceptable excipient.

38. The formulation of Claim 35, wherein said formulation comprises a pharmaceutically acceptable excipient selected from the group consisting of biocompatible polymers,

other polymeric matrices, capsules, microcapsules, microparticles, bolus preparations, osmotic pumps, diffusion devices, liposomes, lipospheres, viral vectors and transdermal delivery systems.

39. The method of Claim 35, wherein said TGFβ-regulating agent is an isolated TGFβ1 protein.

40. The method of Claim 35, wherein said TGFβ-regulating agent is an isolated nucleic acid molecule encoding a TGFβ1 protein, wherein said nucleic acid molecule is operatively linked to a transcription control sequence.

41. The method of Claim 40, wherein said isolated nucleic acid molecule is administered to said mammal complexed with a liposome delivery vehicle.

42. The method of Claim 40, wherein said isolated nucleic acid molecule is administered to said mammal in a viral vector delivery vehicle.

43. The method of Claim 42, wherein said viral vector delivery vehicle is from adenovirus.

44. The method of Claim 40, wherein said isolated nucleic acid molecule, when administered to said mammal, is expressed in cells of said mammal.

45. The method of Claim 35, wherein said TGFβ-regulating agent is an antibody.

46. The method of Claim 35, wherein said TGFβ-regulating agent is an antisense oligonucleotide which hybridizes under stringent hybridization conditions to TGFβ.

47. The method of Claim 35, wherein said TGFβ-regulating agent is a TGFβ-specific ribozyme.

48. The method of Claim 35, wherein said TGFβ-regulating agent is a TGFβ receptor agonist.

49. The method of Claim 35, wherein said TGFβ-regulating agent is a TGFβ receptor antagonist.